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NEWS 1		Web Page for STN Seminar Schedule - N. America
NEWS 2	JUL 02	LMEDLINE coverage updated
NEWS 3	JUL 02	SCISEARCH enhanced with complete author names
NEWS 4	JUL 02	CHEMCATS accession numbers revised
NEWS 5	JUL 02	CA/CAplus enhanced with utility model patents from China
NEWS 6	JUL 16	CAplus enhanced with French and German abstracts
NEWS 7	JUL 18	CA/CAplus patent coverage enhanced
NEWS 8	JUL 26	USPATFULL/USPAT2 enhanced with IPC reclassification
NEWS 9	JUL 30	USGENE now available on STN
NEWS 10	AUG 06	CAS REGISTRY enhanced with new experimental property tags
NEWS 11	AUG 06	FSTA enhanced with new thesaurus edition
NEWS 12	AUG 13	CA/CAplus enhanced with additional kind codes for granted patents
NEWS 13	AUG 20	CA/CAplus enhanced with CAS indexing in pre-1907 records
NEWS 14	AUG 27	Full-text patent databases enhanced with predefined patent family display formats from INPADOCDB
NEWS 15	AUG 27	USPATOLD now available on STN
NEWS 16	AUG 28	CAS REGISTRY enhanced with additional experimental spectral property data
NEWS 17	SEP 07	STN AnaVist, Version 2.0, now available with Derwent World Patents Index
NEWS 18	SEP 13	FORIS renamed to SOFIS
NEWS 19	SEP 13	INPADOCDB enhanced with monthly SDI frequency
NEWS 20	SEP 17	CA/CAplus enhanced with printed CA page images from 1967-1998
NEWS 21	SEP 17	CAplus coverage extended to include traditional medicine patents
NEWS 22	SEP 24	EMBASE, EMBAL, and LEMBASE reloaded with enhancements
NEWS 23	OCT 02	CA/CAplus enhanced with pre-1907 records from Chemisches Zentralblatt
NEWS 24	OCT 19	BEILSTEIN updated with new compounds

NEWS EXPRESS 19 SEPTEMBER 2007: CURRENT WINDOWS VERSION IS V8.2, CURRENT MACINTOSH VERSION IS V6.0c(ENG) AND V6.0Jc(JP), AND CURRENT DISCOVER FILE IS DATED 19 SEPTEMBER 2007.

NEWS HOURS	STN Operating Hours Plus Help Desk Availability
NEWS LOGIN	Welcome Banner and News Items
NEWS IPC8	For general information regarding STN implementation of IPC 8

Enter NEWS followed by the item number or name to see news on that specific topic.

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* * * * * * * * * * * * * * * * STN  Columbus  * * * * * * * * * * * * * * * *

*IMSDRUGNEWS  - IMS Drug News 1991-present

  * The files listed above are temporarily unavailable.

FILE 'HOME' ENTERED AT 12:54:27 ON 20 OCT 2007

=> File .Gerry2MBCE
COST IN U.S. DOLLARS                                    SINCE FILE      TOTAL
                                                           ENTRY    SESSION
FULL ESTIMATED COST                                         0.21       0.21

FILE 'MEDLINE' ENTERED AT 12:54:54 ON 20 OCT 2007

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=> S Activated(2A)alpha2-microglobulin (L)Fatty(A)acid AND pd<=20031230
   1 FILES SEARCHED...
L1            0 ACTIVATED(2A) ALPHA2-MICROGLOBULIN (L) FATTY(A) ACID AND PD<=200
                31230

=> Log off h
 SESSION WILL BE HELD FOR 120 MINUTES
STN INTERNATIONAL SESSION SUSPENDED AT 12:57:48 ON 20 OCT 2007

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SESSION RESUMED IN FILE 'MEDLINE, BIOSIS, CAPLUS, EMBASE'
AT 12:59:28 ON 20 OCT 2007
FILE 'MEDLINE' ENTERED AT 12:59:28 ON 20 OCT 2007
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                                                           ENTRY    SESSION
FULL ESTIMATED COST                                        15.71      15.92

=> D Hist

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     FILE 'MEDLINE, BIOSIS, CAPLUS, EMBASE' ENTERED AT 12:54:54 ON 20 OCT 2007
L1             0 S ACTIVATED(2A)ALPHA2-MICROGLOBULIN (L)FATTY(A)ACID AND PD<=200

=> S  ACTIVATED(2A)ALPHA2-MACROGLOBULIN (L)FATTY(A)ACID AND PD<=20031230
   2 FILES SEARCHED...
L2             2 ACTIVATED(2A) ALPHA2-MACROGLOBULIN (L) FATTY(A) ACID AND PD<=200
                 31230

=> D Ti L2 1-2

L2   ANSWER 1 OF 2     MEDLINE on STN
TI   Fatty acids modulate transforming growth factor-beta activity and plasma
     clearance.

L2   ANSWER 2 OF 2  BIOSIS  COPYRIGHT (c) 2007 The Thomson Corporation  on STN
TI   Adipocyte low density lipoprotein receptor-related protein gene expression
     and function is regulated by peroxisome proliferator-activated receptor
     gamma.


=> D ibib abs l2 2

L2   ANSWER 2 OF 2  BIOSIS  COPYRIGHT (c) 2007 The Thomson Corporation  on STN
ACCESSION NUMBER:    2003:243873  BIOSIS
DOCUMENT NUMBER:     PREV200300243873
TITLE:               Adipocyte low density lipoprotein receptor-related protein
                     gene expression and function is regulated by peroxisome
                     proliferator-activated receptor gamma.
AUTHOR(S):           Gauthier, Andre; Vassiliou, Gerard; Benoist, Fabienne;
                     McPherson, Ruth [Reprint Author]
CORPORATE SOURCE:    University of Ottawa, 40 Ruskin St., Rm. H441, Ottawa, K1Y
                     4W7, Canada
                     rmcpherson@ottawaheart.ca
SOURCE:              Journal of Biological Chemistry, (April 4 2003)
                     Vol. 278, No. 14, pp. 11945-11953. print.
                     CODEN: JBCHA3. ISSN: 0021-9258.
DOCUMENT TYPE:       Article
LANGUAGE:            English
ENTRY DATE:          Entered STN: 21 May 2003
                     Last Updated on STN: 21 May 2003
AB   The alpha2-macroglobulin receptor/low density lipoprotein receptor-related
     protein (LRP) is a large multifunctional receptor that interacts with a
     variety of molecules.  It is implicated in biologically important
     processes such as lipoprotein metabolism, neurological function, tissue
     remodeling, protease complex clearance, and cell signal transduction.
     However, the regulation of LRP gene expression remains largely unknown.
     In this study, we have analyzed 2 kb of the 5'-flanking region of the LRP
     gene and identified a predicted peroxisome proliferator response element
     (PPRE) from -1185 to -1173.  Peroxisome proliferator-activated receptor
     gamma (PPARgamma) ligands such as fatty acids and
     rosiglitazone increased functional cell surface LRP by 1.5-2.0-fold in
     primary human adipocytes and in the SW872 human liposarcoma cell line as
     assessed by activated alpha2-macroglobulin
     binding and degradation.  These agents were found to increase LRP
     transcription.  Gel shift analysis of the putative PPRE demonstrated
     direct binding of PPARgamma/retinoid X receptor alpha heterodimers to the
     PPRE in the LRP gene.  Furthermore, these heterodimers could no longer
     interact with a mutated PPRE probe.  The isolated promoter was functional
     in SW872 cells, and its activity was increased by 1.5-fold with the
     addition of rosiglitazone.  Furthermore, the isolated response element was
     similarly responsive to rosiglitazone when placed upstream of an ideal
     promoter.  Mutagenesis of the predicted PPRE abolished the ability of this
     construct to respond to rosiglitazone.  These data demonstrate that
     fatty acids and rosiglitazone directly stimulate
     transcription of the LRP gene through activation of PPARgamma and increase
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functional LRP expression.

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FILE 'MEDLINE' ENTERED AT 14:49:19 ON 20 OCT 2007
FILE 'BIOSIS' ENTERED AT 14:49:19 ON 20 OCT 2007
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FILE 'CAPLUS' ENTERED AT 14:49:19 ON 20 OCT 2007
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FILE 'EMBASE' ENTERED AT 14:49:19 ON 20 OCT 2007
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COST IN U.S. DOLLARS	SINCE FILE ENTRY	TOTAL SESSION
FULL ESTIMATED COST	34.14	34.35

=> D Hist

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FILE 'MEDLINE, BIOSIS, CAPLUS, EMBASE' ENTERED AT 12:54:54 ON 20 OCT 2007
L1 0 S ACTIVATED(2A)ALPHA2-MICROGLOBULIN (L)FATTY(A)ACID AND PD<=200
L2 2 S ACTIVATED(2A)ALPHA2-MACROGLOBULIN (L)FATTY(A)ACID AND PD<=20

=> S Fatty(2A)Acid (S) TGFbeta AND pd<=20031230
2 FILES SEARCHED...
L3 5 FATTY(2A) ACID (S) TGFBETA AND PD<=20031230

=> D ibib abs L3 1-5

L3 ANSWER 1 OF 5 MEDLINE on STN
ACCESSION NUMBER: 2001301806 MEDLINE
DOCUMENT NUMBER: PubMed ID: 11246816
TITLE: Fat cell function and fibrinolysis.
AUTHOR: Alessi M C; Morange P; Juhan-Vague I
CORPORATE SOURCE: Laboratory of Hematology, Faculty of Medicine, CHU Timone, Marseille, France.
SOURCE: Hormone and metabolic research. Hormon- und Stoffwechselforschung. Hormones et metabolisme, (2000 Nov-Dec) Vol. 32, No. 11-12, pp. 504-8. Ref: 62
Journal code: 0177722. ISSN: 0018-5043.
PUB. COUNTRY: Germany: Germany, Federal Republic of
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200105
ENTRY DATE: Entered STN: 4 Jun 2001
Last Updated on STN: 4 Jun 2001

Entered Medline: 31 May 2001

AB Plasminogen activator inhibitor-1 (PAI-1) is a specific inhibitor of plasminogen activators and may be the principal regulator of plasminogen activation in vivo. PAI-1 levels are elevated in insulin-resistant subjects and are associated with an increased risk of atherothrombosis. After adjustment for metabolic parameters, increased PAI-1 levels were no longer considered as a cardiovascular risk factor. The mechanisms underlying the strong association between PAI-1 levels and the metabolic disturbances found in insulin resistance are still not understood. Several studies have suggested that visceral adipose tissue may be a major source of PAl-1. Accordingly, adipose tissue PAI-1 production particularly that from visceral fat, was found to be elevated in obese human subjects. Within human adipose tissue, stromal cells appear to be the main cells involved in PAI-1 synthesis. This review discusses the potential mechanisms linking adipose tissue to plasma PAI-1 levels such as the intervention of cytokines (TNFalpha and TGFbeta), free fatty acids and hormones (insulin and glucocorticoids). Moreover alteration of adipose tissue cellular composition induced by the modulation of PAI-1 expression opens a novel field of interest.

L3 ANSWER 2 OF 5 MEDLINE on STN
ACCESSION NUMBER: 2001192510 MEDLINE
DOCUMENT NUMBER: PubMed ID: 11204450
TITLE: Free radicals, cytokines and nitric oxide in cardiac failure and myocardial infarction.
AUTHOR: Das U N
CORPORATE SOURCE: EFA Sciences LLC, Norwood, MA 02062, USA.
SOURCE: Molecular and cellular biochemistry, (2000 Dec) Vol. 215, No. 1-2, pp. 145-52. Ref: 60
Journal code: 0364456. ISSN: 0300-8177.
PUB. COUNTRY: Netherlands
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200104
ENTRY DATE: Entered STN: 10 Apr 2001
Last Updated on STN: 10 Apr 2001
Entered Medline: 5 Apr 2001

AB Myocardial infarction is the most common cause of congestive cardiac failure. Free radicals, cytokines, nitric oxide (NO) and antioxidants play a major role both in atherosclerosis and myocardial damage and preservation. In the early stages of atherosclerosis, neutrophils and monocytes infiltrate the intima and generate free radicals which damage the endothelial cells. As a result, production of NO and prostacyclin by the endothelial cells declines, which have cardioprotective actions. This also has relevance to the beneficial action of aspirin since, it can modulate both prostanoid and L-arginine-NO systems and NF-kB translocation. In both acute myocardial infarction and chronic congestive cardiac failure, the plasma levels of various inflammatory mediators such as interleukins and tumour necrosis factor-alpha (TNFalpha) are elevated. TNFalpha, produced by the inflammatory cells and the myocardium, can suppress myocardial contractility and induce the production of free radicals, which in turn can further damage the myocardium. Transforming growth factor beta (TGFbeta), polyunsaturated fatty acids and the glucose-insulin-potassium regimen can antagonize the harmful actions of TNFalpha and protect the myocardium. This explains why efforts made to reduce the levels of pro-inflammatory cytokines have beneficial action and preserve the myocardium.

L3 ANSWER 3 OF 5 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on STN
ACCESSION NUMBER: 2001:164234 BIOSIS
DOCUMENT NUMBER: PREV200100164234
TITLE: Fat cell function and fibrinolysis.
AUTHOR(S): Alessi, M. C.; Morange, P.; Juhan-Vague, I. [Reprint

author]
CORPORATE SOURCE: Laboratory of Hematology, Faculty of Medicine, CHU Timone, 13385, Marseille Cedex 5, France
ijuhan@ap-hm.fr
SOURCE: Hormone and Metabolic Research, (November-December, 2000) Vol. 32, No. 11-12, pp. 504-508. print.
CODEN: HMMRA2. ISSN: 0018-5043.
DOCUMENT TYPE: Article
General Review; (Literature Review)
LANGUAGE: English
ENTRY DATE: Entered STN: 4 Apr 2001
Last Updated on STN: 15 Feb 2002

AB Plasminogen activator inhibitor-1 (PAI-1) is a specific inhibitor of plasminogen activators and may be the principal regulator of plasminogen activation in vivo. PAI-1 levels are elevated in insulin-resistant subjects and are associated with an increased risk of atherothrombosis. After adjustment for metabolic parameters, increased PAI-1 levels were no longer considered as a cardiovascular risk factor. The mechanisms underlying the strong association between PAI-1 levels and the metabolic disturbances found in insulin resistance are still not understood. Several studies have suggested that visceral adipose tissue may be a major source of PAI-1. Accordingly, adipose tissue PAI-1 production particularly that from visceral fat, was found to be elevated in obese human subjects. Within human adipose tissue, stromal cells appear to be the main cells involved in PAI-1 synthesis. This review discusses the potential mechanisms linking adipose tissue to plasma PAI-1 levels such as the intervention of cytokines (TNFalpha and TGFbeta), free fatty acids and hormones (insulin and glucocorticoids). Moreover alteration of adipose tissue cellular composition induced by the modulation of PAI-1 expression opens a novel field of interest.

L3 ANSWER 4 OF 5 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on STN
ACCESSION NUMBER: 2001:130453 BIOSIS
DOCUMENT NUMBER: PREV200100130453
TITLE: Free radicals, cytokines and nitric oxide in cardiac failure and myocardial infarction.
AUTHOR(S): Das, U. N. [Reprint author]
CORPORATE SOURCE: EFA Sciences LLC, Providence Highway, Suite No. 266, Norwood, MA, 02062, USA
SOURCE: Molecular and Cellular Biochemistry, (December, 2000) Vol. 215, No. 1-2, pp. 145-152. print.
CODEN: MCBIB8. ISSN: 0300-8177.
DOCUMENT TYPE: Article
General Review; (Literature Review)
LANGUAGE: English
ENTRY DATE: Entered STN: 14 Mar 2001
Last Updated on STN: 15 Feb 2002

AB Myocardial infarction is the most common cause of congestive cardiac failure. Free radicals, cytokines, nitric oxide (NO) and antioxidants play a major role both in atherosclerosis and myocardial damage and preservation. In the early stages of atherosclerosis, neutrophils and monocytes infiltrate the intima and generate free radicals which damage the endothelial cells. As a result, production of NO and prostacyclin by the endothelial cells declines, which have cardioprotective actions. This also has relevance to the beneficial action of aspirin since, it can modulate both prostanoid and L-arginine-NO systems and NF-kB translocation. In both acute myocardial infarction and chronic congestive cardiac failure, the plasma levels of various inflammatory mediators such as interleukins and tumour necrosis factor-alpha (TNFalpha) are elevated. TNFalpha, produced by the inflammatory cells and the myocardium, can suppress myocardial contractility and induce the production of free radicals, which in turn can further damage the myocardium. Transforming growth factor beta (TGFbeta), polyunsaturated fatty acids and the glucose-insulin-potassium regimen can antagonize the harmful actions of TNFalpha and protect the myocardium. This explains why

efforts made to reduce the levels of pro-inflammatory cytokines have beneficial action and preserve the myocardium.

L3 ANSWER 5 OF 5 CAPLUS COPYRIGHT 2007 ACS on STN
ACCESSION NUMBER: 2003:606429 CAPLUS
DOCUMENT NUMBER: 139:240515
TITLE: Fatty acids modulate transforming growth factor-β activity and plasma clearance
AUTHOR(S): Ling, Thai-Yen; Huang, Yen-Hua; Lai, Ming-Chih; Huang, Shuan Shian; Huang, Jung San
CORPORATE SOURCE: Inst. of Biomed. Sci., Acad. Sinica, Taipei, Taiwan
SOURCE: FASEB Journal (2003), 17(11), 1559-1561, 10.1096/fj.02-1063fje
CODEN: FAJOEC; ISSN: 0892-6638
PUBLISHER: Federation of American Societies for Experimental Biology
DOCUMENT TYPE: Journal
LANGUAGE: English
AB The activity and plasma clearance of transforming growth factor (TGF)-β are known to be regulated by activated α2-macroglobulin (α2M*). This has been implicated in pathophysiol. processes, but no small mol. compds. have been reported to modulate TGF-β activity by affecting the interaction of TGF-β and α2M*. Here, we demonstrate that fatty acids are capable of inhibiting complex formation of TGF-β isoforms and α2M* as demonstrated by nondenaturing and SDS-PAGE. This is dependent on carbon chain length (C20, C18, C16, C14 > C12 > C10), degree of unsatn. (polyunsatd. > saturated), and TGF-β isoforms (TGF-β1 > TGF-β2 > TGF-β3). Arachidonic acid, which is one of the most potent inhibitors, is also capable of dissociating TGF-β-α2M* complexes, but higher concns. are required. Arachidonic acid appears to inhibit TGF-β-α2M* complex formation by binding specifically to α2M* as demonstrated by gel filtration chromatog. Arachidonic acid reverses the inhibitory effect of α2M* on TGF-β binding, TGF-β-induced growth inhibition, and TGF-β-induced transcriptional activation in mink lung epithelial cells and affects plasma clearance of TGF-β-α2M* complexes in mice. These results show that fatty acids are effective modulators of TGF-β activity and plasma clearance and may be useful in treating human diseases through their effects on the interaction of TGF-β and α2M*.
REFERENCE COUNT: 39 THERE ARE 39 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

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